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Obs 3-5, 8, 9, 14-17

L13: Entry 5 of 5 File: DWPI Jan 21, 1992

DERWENT-ACC-NO: 1992-079707
DERWENT-WEEK: 199210

TITLE: Virus inhibition by admin. of hydroxamic acid deriv. - esp. inhibiting viruses dependent on reverse transcriptase replication e.g. human immunodeficiency-and retrovirus and hepatitis-B

INVENTOR: EPSTEIN, J S; HEWLETT, I K ; TABOR, E

PRIORITY-DATA: 1991US-0672577 (March 20, 1991)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 7672577 A	January 21, 1992	N/A	000	N/A
AU 9216726 A	October 21, 1992	N/A	000	A61K031/19
WO 9216200 A1	October 1, 1992	E	017	A61K031/19

INT-CL (IPC): A61K 0/01; A61K 31/19

ABSTRACTED-PUB-NO: US 7672577A
BASIC-ABSTRACT:

Inhibiting comprising administering a hydroxamic acid deriv. (I) of low toxicity.

USE - HIV e.g. HIV-1 and HIV-2 retroviruses e.g. HTLV-I and HTLV-II and hepatitis B. (I) are admin. as soon as positive HIV response has been observed or after the symptoms i.e. AIDS or ARC have appeared. Admin. is i.v. i.p, i,m, s.c. i.d. pref. i.v. or i,m or p.o, at daily doses of 0.1-200 (1-10) mg/kg. Deferoxamine is also active against human hepatocellular carcinoma, human neuroblastoma, human lymphoma and human leukamia.

1, 2, ~~3~~, 10, 11, 12, 13 103

increasing retab. of histamine

stim. cAMP prod.

myeloma

undergoing self-life

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L14: Entry 5 of 18 File: DWPI Mar 14, 2000

DERWENT-ACC-NO: 2000-246182
DERWENT-WEEK: 200021

TITLE: Oral dosage forms e.g. coated caplets for controlled release of tacrine over twenty four hours, useful for treating neurological diseases, particularly Alzheimer's disease

INVENTOR: CHILDERS, J D; GUITTARD, G V ; GUMUCIO, F E ; KIDNEY, D J ; WONG, P S

PRIORITY-DATA: 1997US-0892995 (July 15, 1997), 1994US-0266045 (June 27, 1994)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 6036973 A	March 14, 2000	N/A	016	A61K009/00

INT-CL (IPC): A61K 9/00; A61K 31/13; A61K 31/135

ABSTRACTED-PUB-NO: US 6036973A
BASIC-ABSTRACT:

NOVELTY - Oral dosage forms for treating neurological diseases, particularly Alzheimer's disease, provide pulsed release and extended release of tacrine.

DETAILED DESCRIPTION - Methods of treating a neurological disease comprise oral administration of:

(a) tacrine such that 20-50 mg is administered over 0-2 hours, 60-120 mg over 0-8 hours, 110-170 mg over 0-14 hours and 170-200 mg over 0-24 hours, together with another active agent selected from aniracetam, bifemelane, phosphatidylserine, pramiracetam, physostigmine, fampridine, linopirdine, selegiline, nimodipine, estrogen, propentofylline, alpha -tocopherol, aminopyridine, cytisine, 1-hydroxy-tacrine and donepezil; or

(b) a pulsed release dose of tacrine, together with selegiline, alpha -tocopherol, 1-hydroxy-tacrine or donepezil, an extended release dose of tacrine with a release pattern of 10-25 % after 0-2 hours, 30-60 % after 0-8 hours, 55-85 % after 0-14 hours and greater than 85 % after 0-24 hours, and an active agent as described in (a).

INDEPENDENT CLAIMS are included for:

(i) a dosage form for treating a neurological disease, comprising tacrine and a salt that is administered instantly or in up to 30 minutes, with one of selegiline, alpha -tocopherol, 1-hydroxy- tacrine or donepezil, and an extended-release dose comprising tacrine and a salt that is administered from 0.5-24 hours and another active ingredient as listed in (a);

(ii) caplets for oral administration in the treatment of Alzheimer's disease comprising a composition (c) of 100 ng to 500 mg tacrine and an active agent listed in (a), an expandable composition that imbibes fluid and increases in volume for displacing (c) from the caplet, a wall, permeable to fluid and impermeable to (c), surrounding the composition and having a passageway for delivery of the composition and a coating on the surface of the caplet comprising selegiline, tacrine, alpha -tocopherol, 1-hydroxy tacrine or donepezil;

(iii) a dosage form for delivery to the gastrointestinal environment in treatment of Alzheimer's disease comprising (c), a wall as described in (ii), but which is permeable to gastrointestinal fluid and a coating as described in (ii), where the dosage is delivered over a 24 hour period;

(iv) an osmotic dosage form for delivering tacrine to the gastrointestinal tract in treatment of Alzheimer's disease comprising a tacrine composition containing 108 mg tacrine hydrochloride, 154.80 mg sodium carboxymethyl cellulose (sodium CMC), 79.20 mg sorbitol, 14.40 mg polyvinyl pyrrolidone (PVP) and 3.60 mg magnesium stearate, a displacement composition containing 84.60 mg sodium CMC, 43.20 mg sodium chloride, 7.20 g hydroxypropylcellulose, 7.20 g hydroxypropylmethylcellul- ose and 0.36 mg magnesium stearate, which develops an osmotic pressure greater than that of the gastrointestinal tract and a wall surrounding the tacrine and displacement compositions comprising an 88:12 by weight mixture of cellulose acetate and polyethyleneglycol, and which has an exit passageway for delivering tacrine over 24 hours;

(v) a dosage form for treatment of Alzheimer's disease comprising a composition of 34 % tacrine, 57 % mannitol, 3 % HPMC, 1 % non-crosslinked PVP, 3 % crosslinked PVP and 1 % magnesium stearate, surrounded by a wall comprising 95 % cellulose acetate and 5 % polyethylene glycol, having an exit and optionally coated with 80 % tacrine, 18 % HPMC and 2 % polyethylene glycol;

(vi) a dosage form for treating Alzheimer's disease comprising (c), 2-60 wt.% of an osmotically active agent, 0.25-15 wt.% of a PVP, 0-20 wt.% of a cellulose ether, and 0.01-10 wt.% of a lubricant and a wall permeable to fluid, coated as in (ii) and with an exit to deliver tacrine over 24 hours; and

(vii) a caplet for treatment of Alzheimer's disease comprising (c) (an additional ingredient is a tacrine salt), a hydrophilic sub coat surrounding (c), a coating as described in (ii)

weighing 1-225 mg, a semipermeable wall surrounding the sub coat and a passageway for delivering (c).

ACTIVITY - Nootropic; Neuroprotective.

MECHANISM OF ACTION - None given.

USE - For treating neurological diseases, particularly Alzheimer's disease (claimed).

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L14: Entry 11 of 18 File: DWPI Nov 25, 1997

DERWENT-ACC-NO: 1998-059080
DERWENT-WEEK: 199806

TITLE: Acetyl:choline neuro:transmitter enhancer - contains selegiline or its salt

PRIORITY-DATA: 1996JP-0158796 (May 15, 1996)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 09301857 A	November 25, 1997	N/A	005	A61K031/135

INT-CL (IPC): A61K 31/135

ABSTRACTED-PUB-NO: JP09301857A
BASIC-ABSTRACT:

Acetylcholine series neurotransmitter enhancer contains selegiline or pharmacologically acceptable salt.

USE - The acetylcholine series neurotransmitter enhancer can modulatory enhance neurotransmitter by acetylcholine between synapse through different mechanism than conventional acetylcholine esterase inhibition mechanism. It is used esp. in ageing, Alzheimer's disease, Parkinsonian dementia or HIV dementia correlated to dysfunction or decidual function of acetylcholine series nerve, and/or via sigma-receptor

ADVANTAGE - Selegiline has affinity to sigma-receptor, induces membrane potential of acetylcholine nerve therethrough, enhances exocytosis in synapse within the limits of side-effects since the action is quite modulatory secondary reaction depended on membrane potential via receptor, and specifically acts on Meynert's basal ganglia-frontal cortex referred cholinergic system which has been recognised as common responsible lesion of disease accompanied with cognition injury.

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L14: Entry 12 of 18 File: DWPI May 29, 2001

DERWENT-ACC-NO: 1997-479861
DERWENT-WEEK: 200132

TITLE: Administration of selegiline to a patient - to treat or prevent peripheral neuropathy caused by e.g. chemotherapeutic agent

INVENTOR: BOBOTAS, G

PRIORITY-DATA: 1996US-0013520 (March 15, 1996), 1999US-0402752 (August 13, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 6239181 B1	May 29, 2001	N/A	000	A61K031/135
WO 9733572 A1	September 18, 1997	E	022	A61K031/135
AU 9723397 A	October 1, 1997	N/A	000	A61K031/135
EP 906090 A1	April 7, 1999	E	000	A61K031/135
CN 1213300 A	April 7, 1999	N/A	000	A61K031/135
JP 11507951 W	July 13, 1999	N/A	024	A61K031/135
AU 709323 B	August 26, 1999	N/A	000	A61K031/135

INT-CL (IPC): A61K 31/135; A61K 31/335; A61K 31/475; A61K 33/24; A61K 45/00

ABSTRACTED-PUB-NO: US 6239181B
BASIC-ABSTRACT:

To treat or prevent peripheral neuropathy caused by a toxic agent, a genetically inherited condition or a systemic disease, selegiline (L-(-)-deprenyl or R-(-)-deprenyl), (I) is administered to reduce or eliminate one or more symptoms.

Also claimed is a method of treatment using (I).

The toxic agents are cisplatin, paclitaxel, vincristine and vinblastin.

USE - The selegiline compound may especially be used to treat cancer, especially concurrently with a chemotherapeutic agent known to have a toxic effect on peripheral nerves.

Preferably the selegiline is administered to a human by a route that avoid its absorption from the gastrointestinal tract.

Transdermal, buccal and sublingual routes are preferred, daily dosage being in the range 0.01-0.15 mg/kg, based upon the weight of free amine.

ADVANTAGE - The method enables individual or cumulative dose of the agent to be maximised without unacceptable severe neurotoxic side-effects to the patient.
ABSTRACTED-PUB-NO:

WO 9733572A EQUIVALENT-ABSTRACTS:

To treat or prevent peripheral neuropathy caused by a toxic agent, a genetically inherited condition or a systemic disease, selegiline (L-(-)-deprenyl or R-(-)-deprenyl), (I) is administered to reduce or eliminate one or more symptoms.

Also claimed is a method of treatment using (I).

The toxic agents are cisplatin, paclitaxel, vincristine and vinblastin.

USE - The selegiline compound may especially be used to treat cancer, especially concurrently with a chemotherapeutic agent known to have a toxic effect on peripheral nerves.

Preferably the selegiline is administered to a human by a route that avoid its absorption from the gastrointestinal tract. Transdermal, buccal and sublingual routes are preferred, daily dosage being in the range 0.01-0.15 mg/kg, based upon the weight of free amine.

ADVANTAGE - The method enables individual or cumulative dose of the agent to be maximised without unacceptable severe neurotoxic side-effects to the patient.